

Chapter 3

Organochlorine Residues and their Effects on Fish and Wildlife of the North American Great Lakes

Introduction

For nearly two centuries the Great Lakes of North America have been the receiving waters for industrial and municipal wastes. Their contamination with persistent and bioaccumulative organochlorine compounds (OCs), including those designated as persistent organic pollutants (POPs) under the Stockholm Convention, has been studied in detail and demonstrated to cause population-level effects on wildlife. The large size of the lakes had led to the commonly held, yet mistaken, belief that it was impossible to contaminate them sufficiently to cause adverse effects. As human populations and industrialization of the Great Lakes basin increased, and the complexity and magnitude of the industries grew, it became apparent that it was indeed possible to contaminate the lakes to the extent that adverse effects would be observed. The release of persistent and bioaccumulative compounds eventually resulted in thresholds for adverse effects being exceeded in a number of wildlife populations.

Literally thousands of OCs can now be found in the tissues of fish and wildlife of the Great Lakes (Figure 3-1). Concentrations of all of the compounds designated for control under the Stockholm Convention have been found at elevated levels in Great Lakes wildlife, and all could have contributed to some of the observed adverse effects in some species. However, several of the POPs were responsible for most of the observed effects. These include the insecticides dieldrin and DDT, the industrial

polychlorinated biphenyls (PCBs), and the byproducts of incineration and chemical production, polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs). Although these POPs may have had adverse impacts on a number of species, their effects have been best documented for a few sentinel species such as lake trout (*Salvelinus namaycush*) (Giesy and Snyder, 1998), bald eagles (*Haliaeetus leucocephalus*) (Bowerman et al., 1995, 1998), colonial fish-eating water birds such as the cormorant (*Phalacrocorax auritus*) and Caspian tern (*Sterna caspia*) (Giesy et al., 1994a,b), and the mink (*Mustela vison*) (Giesy et al., 1994c; Tillitt et al., 1996).

The experiences in the Great Lakes have resulted in a greater understanding of the potential hazards of releasing persistent, bioaccumulative, toxic compounds into the environment. Many of the pollutants are no longer manufactured or their use is heavily restricted. Concentrations of the most problematic compounds such as DDT and PCBs have declined, but the current rates of decline are very slow, such that it will be a long time before the concentrations in both fish and birds of the Great

Lakes environment reach “background” concentrations (Figures 3-2 to 3-4) (Giesy and Snyder, 1998). The trends for PCBs and DDTs are similar to those for the other Stockholm Convention POPs in both fish and birds. These trends demonstrate several things. First, fish and wildlife became contaminated with POPs resi-

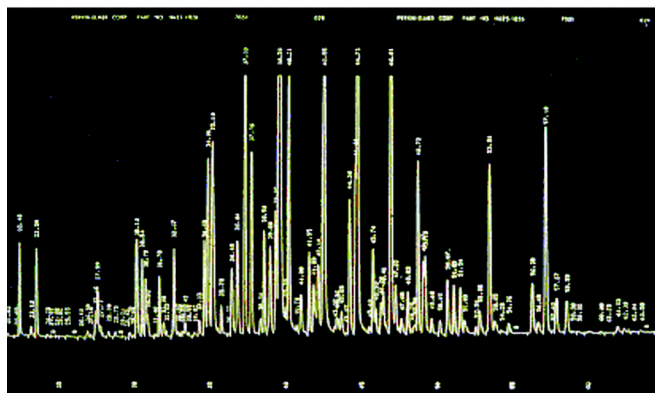


Figure 3-1. Chromatogram showing organochlorine compounds in extract of Great Lakes fish.

Photo: J.P. Giesy

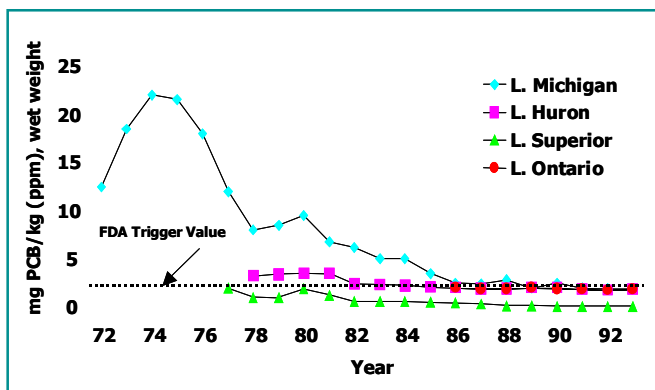


Figure 3-2. Concentrations of PCBs in lake trout from the four uppermost Great Lakes between 1972-1993. Redrawn from MDNR (1994).

dues soon after they were introduced, reaching maximum concentrations in the early 1970s. Second, when use of these compounds was restricted in North America, concentrations began decreasing immediately and decreased to concentrations at or near the threshold for effects within approximately 20 years. Even though the manufacture of these compounds ceased over 25 years ago, they remain at significant concentrations in wildlife. This lingering effect occurs because concentrations of POPs, once introduced into the environment, take a long time to decrease to nondetectable concentrations. Third, current concentrations in biota are no longer decreasing at the same rate that they once were. This slower rate is caused, in part, by continued input to the lakes from reservoir sources and from long-range atmospheric transport originating in other areas of the world (Giesy and Snyder, 1998; Simcik et al., 1999).

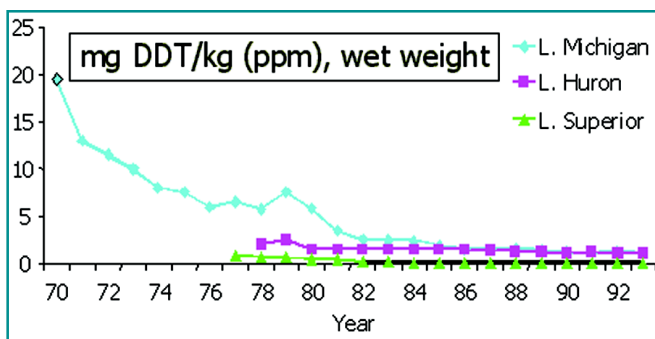


Figure 3-3. Concentrations of total DDTs in lake trout in the three uppermost Great Lakes. Redrawn from MDNR (1994).

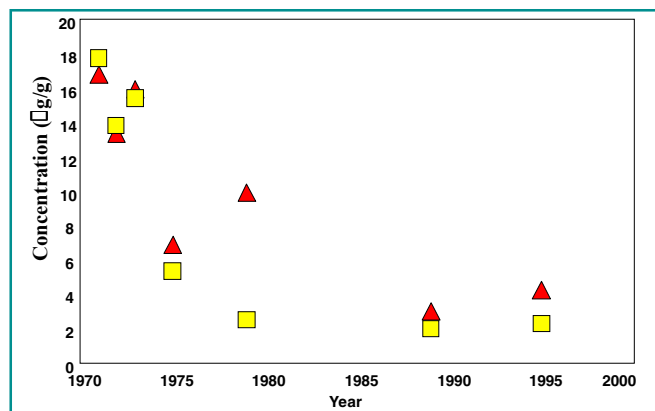


Figure 3-4. Concentrations of PCBs and DDTs in double-crested cormorant eggs collected from the Great Lakes (yellow squares = DDTs; red triangles = PCBs). Data from Environment Canada.

In this report we provide examples of the concentrations of selected POPs in wildlife and the adverse effects that have been caused by exposure to these compounds in the Great Lakes. To do this, we perform a simple risk screening by comparing concentrations of the POPs in tissues or the diets of wildlife with the threshold concentration for adverse effects. This comparison is done by calculating hazard quotients (HQs) (Table 3-1). The HQ can be calculated using tissue concentrations or dietary concentrations. For instance, the concentration of PCBs in fish in the diet of a fish-eating bird can be compared with the dietary No Observable Adverse Effect Level (or concentration) (NOAEL). The NOAEL is the concentration above which effects would be increasingly expected. A HQ value of 10 indicates that the dietary concentration of PCBs is 10-fold greater than the threshold for causing an adverse effect in the piscivorous birds. Said another way, the concentration of PCBs in that species of fish would need to decrease by a factor of 10 before it would not be expected to cause any adverse effects to the birds that ate it. A HQ below 1 indicates that, to the best of our current knowledge, adverse effects would be unlikely to occur.

Fish

Fish from all of the Great Lakes contain measurable concentrations of POPs and many other OCs (Giesy and Snyder, 1998). In general, fish from

Table 3-1. Calculation of Hazard Quotients (HQs)

$$HQ = \frac{\text{Concentration in Fish}}{\text{NOAEL}}$$

HQs were calculated for:

Total PCBs

TCDD-EQ (calculated)

Calculation of HQ assumes animals eat only one fish of interest. This is a conservative or “worst-case” estimate.

Lakes Ontario and Michigan tend to have the greatest concentrations of OCs. Fish from Lake Ontario contain the highest concentrations of the insecticides mirex, DDT, and dieldrin. The lowest concentrations of most persistent, synthetic, chlorinated hydrocarbons are observed in fish from Lake Superior. The relatively high concentrations of toxaphene found in fish from Lake Superior have been the subject of several investigations (Swackhamer et al., 1998; Shanks et al., 1999; Glassmeyer et al., 2000). Concentrations of OCs in fish tissues have decreased by a factor of approximately 25 since maximum concentrations were reached in the lower Great Lakes in the mid-1970s. Although there are differences among species and locations, in general the trends for persistent OCs are either decreasing or stable.

There is considerable difference of opinion in the literature about the extent to which contaminants have affected Great Lakes fish. The types of effects that have been reported in Great Lakes fish include changes in behavior, reduced reproductive success, thyroid enlargement and decreased thyroid hormone content, premature sexual maturation in males, loss of secondary sexual characteristics, lessened plasma gonadotropin and gonadal hormone content, lessened egg fertility, and greater than expected embryo mortality and deformities (Leatherland, 1993). There is strong evidence that the endocrine systems of salmonid fish such as lake trout and chinook salmon are impaired, but at this time it is not clear if these observed imbalances are the result of exposure to POPs.

Populations of several fish species have changed drastically from historical population levels. Many factors have been implicated in these declines, such as fishing, habitat loss, changes in genetic strains, and effects of the sea lamprey, in addition to a likely role for POPs and other pollutants. Historically, the reproductive success of salmonid fish in the Great Lakes was much poorer than that of the same species raised on the Pacific coast of the United States (Giesy et al., 1986; Willford et al. 1991). These adverse effects were often attributed to toxic substances, but it was difficult to demonstrate a cause-effect linkage. Specifically, several fish species have exhibited reproductive deficits that could be caused by exposure to POPs, although isolating the causal agent has proven problematic because the mixtures of chemicals to which the fish are exposed vary over time and location.

Of the many OCs measured in fish and their eggs, the OCs most often implicated in adverse effects are DDTs, PCBs, polychlorinated dibenzo-*p*-dioxins (PCDDs), and polychlorinated dibenzofurans (PCDFs) (Giesy and Snyder, 1998). The highest concentrations of POPs observed in fish eggs were for PCBs (11 mg/kg) and DDT (7 mg/kg). Based on these levels, it was initially hypothesized that DDT and PCBs were most likely responsible for the observed egg mortality and reproductive toxicity. However, although both DDT and PCBs could cause lethality of lake trout eggs and fry in laboratory studies, the concentrations required to cause 30%–50% mortality were as much as 25 times greater than the concentrations observed in the eggs at that time. Thus, although concentrations of DDT observed historically in Great Lakes salmonids were in the range of the thresholds for adverse effects, as determined in laboratory studies, it is unlikely that these concentrations were the major cause of adverse effects seen in the eggs of feral fish from the Great Lakes. Current evidence indicates that PCDD, PCDF, and some of the non- and mono-*ortho*-substituted PCBs are principally responsible for blue-sac syndrome (Figure 3-5) and impaired reproductive performance of salmonid species in the lower Great Lakes, especially for lake trout (Walker and Peterson 1991, 1994a,b; Zabel et al., 1995; Giesy and Snyder, 1998).

Lake Trout

Lake trout in Lake Michigan have not been naturally reproducing successfully for some time. The lake trout populations in both Lakes Michigan and Huron are maintained by stocking programs because natural reproduction of the populations is not sufficient to sustain populations (Willford et al., 1981). Nevertheless, there has been evidence of natural reproduction in Lake Huron (Weber and Clark, 1984). Populations of lake trout have continued to reproduce naturally in Lake Superior, and reproductive success is improving (Curtis, 1990). A number of studies have indicated that the lake trout population recoveries in Lakes Huron and Michigan are related to reduced toxic organic residues in the eggs (see Giesy and Snyder, 1998, for a comprehensive review).

Between 1978 and 1981, annual rearing mortalities in lake trout fry as great as 97% were described for hatchery-reared fish (Mac et al., 1985). Mortality in these studies could not be attributed to disease or nutrition, and was characterized by erratic swimming behaviors and loss of equilibrium prior to death (swim-up syndrome). Poor survival was significantly correlated with the source of eggs and sperm, more so than the quality of the water in which the eggs were reared (Mac et al., 1985). In addition, a number of the adult lake trout produced fry that developed “blue sac” syndrome. This syndrome presents itself as an edematous condition that results in fluid filling the yolk sac and results in a bluish color.

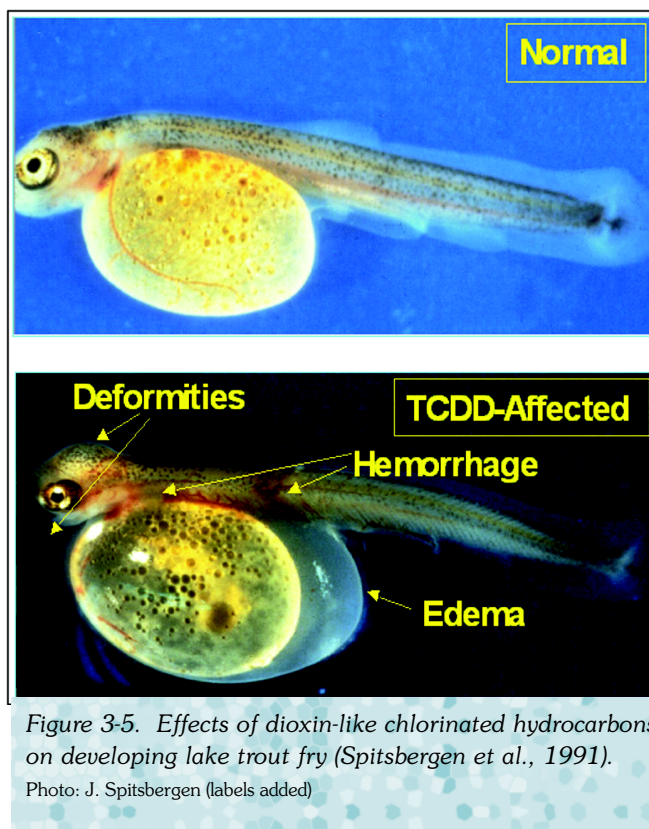


Figure 3-5. Effects of dioxin-like chlorinated hydrocarbons on developing lake trout fry (Spitsbergen et al., 1991).

Photo: J. Spitsbergen (labels added)

Studies during the early 1980s by scientists at the U.S. Fish and Wildlife Service (FWS) research laboratory in Ann Arbor, Michigan, indicated that the most likely cause of poor reproductive success was toxic chemicals (Mac et al., 1985; Willford et al., 1981; Giesy and Snyder, 1998). Their analyses identified 167 chlorinated hydrocarbons in fish, although many more have been identified since. FWS researchers considered it unlikely that a correlation could be established between specific chemicals and effects because

of the large number of compounds observed in fish. They believed contaminants must be involved for the following reasons:

1. Mortality was restricted to lake trout from southern Lake Michigan, which was also the area where the lake trout contained the greatest concentrations of PCBs and DDT. The mortality rate of fry from Lake Superior was small and that lake had the lowest concentrations of PCBs and DDT.
2. Mortality occurred during the swim-up stage of development, during which time fry were most sensitive to the toxic effects of chemicals.
3. The syndrome reached a maximum effect in both lake trout and chinook salmon populations at the same time.

Thus, it was thought that DDT and PCBs were most likely responsible for the observed toxicity. However, no blue-sac disease, the syndrome ob-

served in the fry hatched from eggs of feral females, was observed in laboratory studies of the effects of DDT or PCBs. This finding suggested that the complex mixture of contaminants in fish, and the cause of blue-sac disease, had not been completely identified or quantified.

Further studies on lake trout in the lower Great Lakes demonstrated the link between blue-sac disease/impaired reproductive performance and the levels of PCDD, PCDF, and some of the non- and mono-*ortho*-substituted PCBs measured as dioxin toxicity equivalents (TEQs) (Walker et al., 1991; Walker and Peterson, 1991, 1994a,b; Cook et al., 1997). The threshold for toxic effects of TEQ on lake trout sac fry is approximately 30-40 ng/kg (LD_{50} ~47-65 ng/kg) wet weight (parts per trillion [ppt]) (Figure 3-6) (Walker et al. 1991; Walker and Peterson, 1994a,b). Toxicity is manifest from 1 week prior to hatching through the sac-fry stage of development (Spitsbergen et al., 1991). Lake trout are among the most sensitive species to dioxin toxicity. Thus, they can be used as sentinels for other species. Current concentrations of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin TEQs remain near the threshold for mortality in lake trout fry (Walker and Peterson, 1994a). By 1988, concentrations of TEQs had decreased to 8 and 29 ppt in Lakes Michigan and Ontario, respectively. These concentrations are less than the threshold of approximately 40 ppt, but only slightly in the case of Lake Ontario (see Giesy and Snyder, 1998, for further details).

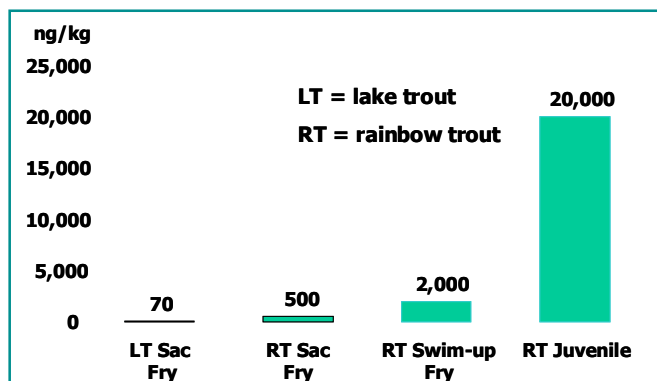


Figure 3-6. LD_{50} concentrations for effects of TEQs on lake trout (redrawn with permission from Walker and Peterson, 1994a).

Although no measurements of historical concentrations of TEQs in fish tissue are available, use of sediment concentrations from a dated sediment core to infer historical concentrations in lake trout indicate that, historically, the threshold concentration would have been exceeded (Cook and Burkhard, 1998). Furthermore, until recently lake trout hatched from females collected from the Great Lakes suffered a relatively high incidence of blue-sac disease. Although it has been reported that this incidence can be caused by bacterial infections (Symula et al., 1990), the edematous condition seen is characteristic of exposure of lake trout to 2,3,7,8-TCDD or structurally similar compounds (Walker and Peterson, 1994a; Cook et al., 1997). Because the dose-response relationship for the dioxin-like compounds in lake trout is so steep, it is likely that the concentrations of dioxin TEQ in the eggs were well above the toxicity threshold for blue-sac syndrome (Guiney et al., 1996) in the recent past. Problematic levels are still reported, where extracts of whole adult lake trout from Lake Michigan continue to lead to lethality when injected into rainbow trout eggs in graded doses (Wright and Tillitt, 1996). The extract, which contained PCDD, PCDF, and PCB congeners, caused yolk-sac edema, cardiofacial deformities, and hemorrhage. All of these symptoms have been observed in Great Lakes fish and can be caused by exposure of fish eggs to TCDD.

In conclusion, it is not possible to determine the actual degree to which POPs have affected lake trout populations in the Great Lakes (Zint et al., 1995). It is likely that dioxin TEQs, primarily from dioxin-like PCB congeners, have caused reproductive impairment of lake trout in the lower lakes, but not Lake Superior. Declines in lake trout populations began in Lakes Ontario, Huron, and Michigan before concentrations of POPs were high enough to drastically reduce reproduction. This observation, along with the fact that "catch per unit effort" generally declines after populations have declined, indicates that initially non-contaminant effects were most likely the cause of the population declines of lake trout. Also, because populations began to decline before sea lamprey numbers were sufficiently high to cause severe population reduc-

tions, the most likely cause of the decline in lake trout populations in the lower lakes was over-exploitation by the commercial fishery. POPs may have played a significant role in delaying reestablishment of lake trout in the lower lakes, but the effects should begin to abate now that concentrations of these compounds have declined to near the threshold for mortality of eggs and fry.

Birds

During the 1960s and 1970s, when the pesticide DDT was being used in the North American environment, populations of several sensitive bird species declined as individuals became unable to successfully incubate eggs because of abnormally thin shells (Cooke, 1973). The eggshell-thinning effect of DDT and its potent, stable, metabolite *p,p'*-DDE in sensitive species is well known, even to the lay public. Indeed, the contributions of DDT to population declines in bird species such as brown pelicans (*Pelecanus occidentalis*), peregrine falcons (*Falco peregrinus*), and bald eagles are probably the most famous incidents in wildlife ecotoxicology. Although many populations worldwide were adversely affected, some of the more notable species that suffered catastrophic declines in the Great Lakes basin included the osprey (*Pandion halieatus*), bald eagle, and many colonial fish-eating birds such as herring gulls (Figure 3-7), common and Caspian terns, and double-crested cormorants (Figure 3-8). In fact, some of these



Figure 3-7. Herring gull colony in Lake Michigan.

Photo: John P. Giesy



Figure 3-8. Double-crested cormorant colony, Lake Huron.

Photo: John P. Giesy

species were almost completely extirpated from the Great Lakes basin. These effects, including declines in populations, have been best documented for colonial, fish-eating water birds (Gilbertson et al. 1991; Peakall and Fox, 1987). Although effects were not restricted to the Great Lakes basin, a number of species in the Great Lakes experienced significant population declines (Bowerman et al., 1995, 1998). Many of these species, such as the double-crested cormorant, have experienced dramatic population increases since DDT was deregistered in the United States and environmental concentrations have declined (Ludwig, 1984; Weseloh and Ewins, 1994).

Bald Eagles

There is no question that bald eagle (Figures 3-9, 3-10) populations declined greatly from historical levels, but the specific reasons for these declines are less clear (Bowerman et al., 1995). The numbers of bald eagles in North America declined greatly after World War II (Grier, 1982; Postupalski, 1985). This decline was particularly acute in the Great Lakes region (Colborn, 1991). Certainly, habitat changes and killing of adults played a role in the population dynamics of bald eagles in the continental United States and Great Lakes basin. However, the greatest effect on bald eagle populations was from DDT residues, specifically the degradation product *p,p'*-DDE, which is known to cause eggshell thinning in eagles (Peakall et al., 1973; Feyk and Giesy, 1998). The effect of



Figure 3-9. Bald eagle egg in eagle nest with habitat in background.

Photo: D. Best/U.S. FWS

DDT residues on bald eagle reproductive success, like many other raptors, is inversely proportional to the dose of p,p' -DDE (Figure 3-11). It is impossible, however, to separate the potential effects of other organochlorine compounds, such as dieldrin, PCBs, and dioxins, all of which accumulated in eagles to concentrations that may have contributed to population-level declines (Wiemeyer et al., 1984, 1993; Giesy et al., 1995; Bowerman et al., 1998). However, due to the specific mechanism of action of p,p' -DDE, it is clear that this compound exceeded concentrations sufficient to cause population-level declines in bald eagles of the Great Lakes basin (Wiemeyer et al., 1984, 1993; Bowerman et al., 1995).

In 1976, just after the use of DDT was canceled in North America, there were approximately 30



Figure 3-10. Bald eagle in flight.

Photo: U.S. FWS

breeding pairs of eagles along the shores of the Great Lakes and 80 and 100 pairs in the interior regions of Michigan and Minnesota, respectively (Figure 3-12). On ceasing the use of DDT in North America, concentrations of p,p' -DDE in the environment, particularly in the diets of fish-eating birds, began to decline. Subsequently, once the concentration had declined below the threshold for population-level effects, populations of eagles began to increase (Grier, 1982; Postupalsky, 1985). The number of breeding pairs has increased steadily since 1976 (Postupalsky, 1985).

Currently, bald eagles nesting along the shoreline of the Great Lakes do not reproduce as well as those nesting on inland bodies of water (Bowerman et al., 1995). Two common measures of the reproductive success of bald eagles are the number of young fledged per occupied nest, referred to as productivity (compared over the total number of nests in a specified region or area), and the reproductive success of each nest (given as the number of young produced in a specific nest, e.g., 0, 1, 2 chicks). Maintenance of a stable bald eagle population requires a productivity of approximately 0.7 chicks/occupied nest/year. A healthy population, capable of exporting excess productivity to colonize other areas, is characterized by a productivity of greater than 1.0. Productivities less than 0.7 are insufficient to replace the loss of adults (Bowerman et al., 1995). The average productivity for bald eagles nesting along the shorelines of the Great Lakes is 0.7, which is just sufficient to maintain a stable population, but not to expand. Any expan-

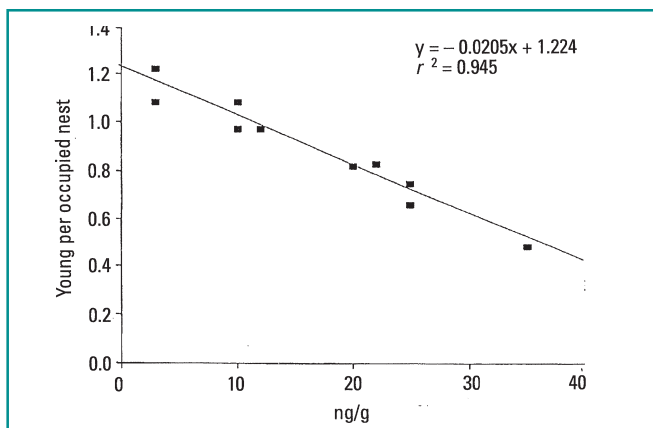


Figure 3-11. Productivity of bald eagles as a function of *p,p'* DDE in eggs (Bowerman et al., 1995).

sion of bald eagle populations along the shoreline of the Great Lakes is due to immigration from other more productive areas. The productivities for the Michigan shoreline on Lakes Michigan and Huron are 0.53 and 0.25, respectively, which is insufficient to maintain stable populations, let alone support any expansion (Figure 3-13). Bald eagles along rivers with anadromous (fish that return to inland streams to breed) populations of salmon from the Great Lakes have a very low productivity of 0.55. The reduced productivities along the shores of the Great Lakes and anadromous salmon streams may be due to a number of factors, including exposure to residual concentrations of *p,p'*-

DDE, but may also be caused by exposure to PCBs, PCDDs, and PCDFs or other compounds (Giesy et al., 1994, Bowerman et al., 1995). In addition, there may be microclimatological effects in some areas and impacts of low food availability (Dykstra et al., 1998). Although it is impossible to know the exact contribution of each of the POPs to eagle reproductive impairment, it is likely that current concentrations of POPs such as PCBs (Figure 3-14) in the Great Lakes are sufficient to cause population-level effects on some subpopulations of bald eagles (Bowerman et al., 1995).

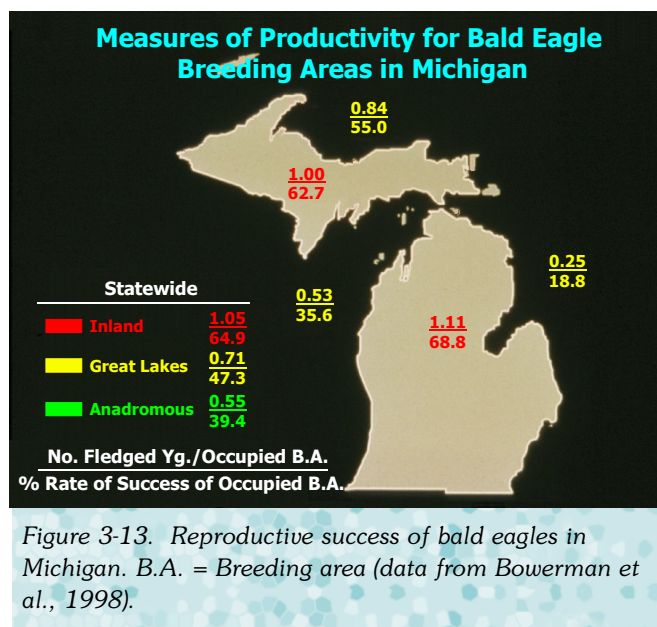


Figure 3-13. Reproductive success of bald eagles in Michigan. B.A. = Breeding area (data from Bowerman et al., 1998).

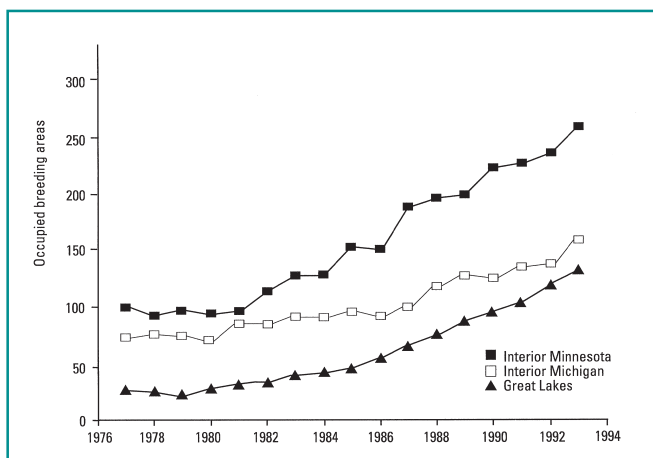


Figure 3-12. Numbers of breeding pairs of bald eagles along the Great Lakes shorelines and in the interiors of Michigan and Minnesota for the period 1977 to 1993 (data from Bowerman et al., 1995).

Colonial Fish-Eating Birds

Current concentrations of PCDD, PCDF, and PCBs in both Great Lakes piscivorous birds and their prey are less than they were in the 1960s and 1970s. Some bird populations, such as double-crested cormorants and herring gulls, have made dramatic recoveries since that time. Populations of other species, such as common and Forster's tern, continue to decline. The concentrations of TEQ in several species appear to be greater than the threshold for discernible, population-level effects at several locations around the Great Lakes (see Giesy et al., 1994b, for a comprehensive review). For instance, subpopulations of double-crested cormorants and Caspian terns in Saginaw Bay and Green

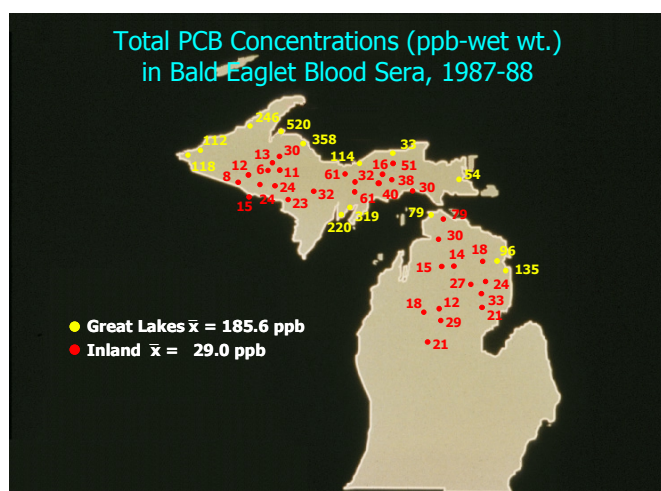


Figure 3-14. Concentrations of PCBs in blood plasma of bald eagles in Michigan (data from Bowerman et al., 1991).

Bay continue to display embryo lethality (Figures 3-15 and 3-16) and abnormally high rates of developmental deformities (Figures 3-17 and 3-18). In general, all of the populations of fish-eating birds from the Great Lakes are displaying symptoms of exposure to chlorinated chemicals at the biochemical level.

PCDDs, PCDFs, and certain structurally similar PCBs have been demonstrated to cause a syndrome referred to as Great Lakes Embryo Mortality Edema and Deformities Syndrome (GLEMEDS) (Table 3-2). This syndrome, which is similar to chick edema disease, results in embryo lethality and developmental deformities in fish-eating birds. Although the degree of expression of GLEMEDS



Figure 3-15. Cormorant eggs. One died while hatching.
Photo: John P. Giesy

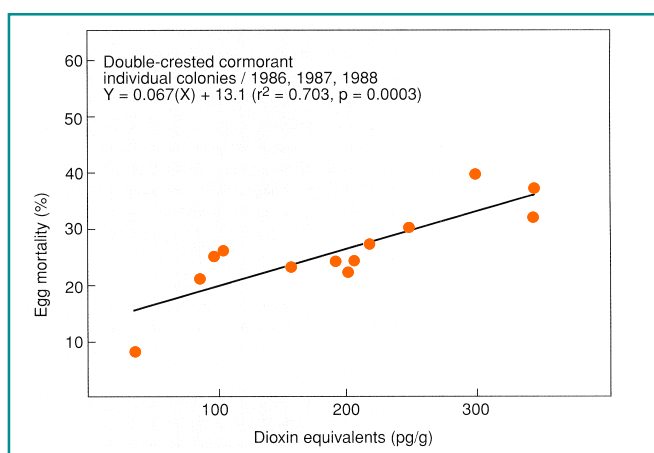


Figure 3-16. Correlation between embryo lethality (egg death) as a function of total dioxin equivalents in double-crested cormorant eggs from the Great Lakes (Giesy et al., 1994a; Tillitt et al., 1992).

has decreased as concentrations of PCDDs, PCDFs, and PCBs have declined, certain species in some locations are still affected. These exposures are still causing lethality and deformities in embryos of all of the populations examined by research groups from the United States and Canada, including those from Michigan State University (extensively reviewed in Giesy et al., 1994a,b). The observed effects are greater than those observed in less contaminated populations not breeding on the Great Lakes, although these effects are translated into biologically significant population-level effects



Figure 3-17. Cormorant with cross-bill malformation.
Photo: John P. Giesy

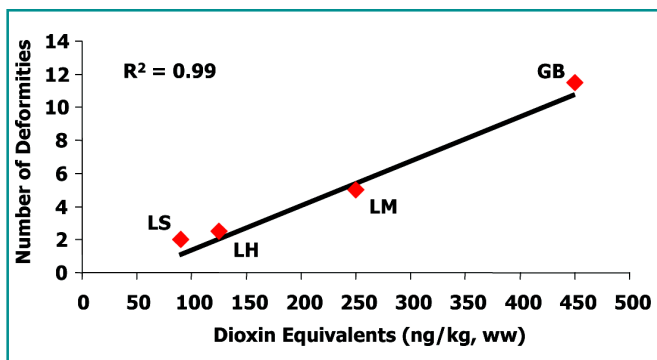


Figure 3-18. Rates of deformities per thousand double-crested cormorant embryos for the Great Lakes (Giesy et al., 1994a). LS–Lake Superior, LM–Lake Michigan, LH–Lake Huron, GB–Green Bay

only in the more contaminated areas, such as Saginaw and Green Bays.

As with fish, the results of laboratory and field studies indicate that the lethality and deformities (Table 3-3) of embryos of fish-eating birds from the Great Lakes are caused by the toxic effects of multiple compounds expressed through the Ah receptor. The use of TEQ as the measurement unit also explains the observed effects better than single measurements of individual compounds (Geisy et al. 1994a). When the current concentrations of PCBs and PCB-derived TEQs in the diets of fish-eating birds of the Great Lakes were compared

Table 3-2. Clinical signs of GLEMEDS

- Embryo lethality
- Liver mixed function oxidase (MFO) induction
- Unabsorbed yolk sacs
- Vitamin A depletion
- Porphyria
- Teratogenesis

Source: Gilbertson et al. (1991).

Table 3-3. Deformities caused by exposure to TEQs

- | | |
|----------------------|-----------------------|
| ● Gastroschisis | ● Hemorrhaging |
| ● Crossed bills | ● Abnormal feathering |
| ● Clubfoot | ● Abnormal eyes |
| ● Dwarfed appendages | ● Hydrocephaly |
| ● Edema/ascites | ● Anencephaly |

Source: Giesy et al. (1994a).

with NOAEL values for these species, based on both laboratory and field studies with these species as well as surrogates, the hazard quotients and exceedence values were greater than 1.0 for all of the species in all of the lakes (Giesy et al., 1994b; 1995). The magnitude of the HQ values varies among species and lakes. Bald eagles are the most exposed and the most sensitive, whereas cormorants are relatively tolerant of the effects of PCB-derived TEQs.

Mammals

In addition to colonial, fish-eating water birds, several other wildlife populations have been reported to be affected by contaminants in Great Lakes fish. Populations of mustelids, including mink (*Mustela vison*) (Figure 3-19) and river otter (*Lutra canadensis*), have declined in regions along the Great Lakes or along rivers that are not blocked by dams, to which fish from the Great Lakes have access (Giesy et al., 1994c). It is difficult to conduct a risk assessment for mink because accurate information on their diets is limited. A number of researchers have reported that feeding fish from the Great Lakes has resulted in adverse effects on ranch mink, and several analyses have been conducted to examine the effects of contaminant compounds (Giesy et al., 1994c; Kannan et al., 2000). In studies feeding Great Lakes fish, mink are simultaneously exposed to a number of synthetic, halogenated compounds, including OC insecticides. Because the concentrations of many of these compounds are intercorrelated, it is difficult to separate their effects and determine which are most likely to have caused adverse effects in populations of wild or ranch mink. As little as 1% Great Lakes fish in the diet of mink is sufficient to cause adverse effects on survival and growth of the kits (Restum et al., 1998; Giesy et al., 1994c); 40% Great Lakes fish in the diet causes mortality of adult female mink (Heaton et al., 1995a,b). When adult female mink were fed 10% Great Lakes fish, the number of surviving kits was significantly reduced (Figure 3-20). Historically, when the concentrations of OC insecticides such as DDTs in the tissue of fish from the Great Lakes were higher, it was concluded that they were unlikely to be the cause of the effects observed



Figure 3-19. Mink in the wild eating fish.

Photo: J. McDonald/Corbis.com

when fish from the Great Lakes were fed to ranch mink. The most likely causes of the observed effects are again the PCDDs, PCDFs, and PCBs. Although it is difficult to determine the exact cause of the observed effects, it is clear that residues present in fish from the Great Lakes can cause significant effects on the survival and reproduction of mink.

Of all the pollutants to which mink have been exposed, PCBs seem to have had the greatest impact. Mink are one of the most sensitive organisms to the effects of PCBs. In an attempt to determine if current concentrations of PCBs represent a risk to mink, hazard quotients were calculated assuming that mink ate only Great Lakes fish in their diet (Heaton et al., 1995a,b). Hazard quotients for PCBs in fishes from the Great Lakes

ranged from a minimum of 6.4 to a maximum of 83. Percent allowable consumption values for feeding Great Lakes fish to mink were all less than 100%, and ranged from as little as 1.2% to as much as 19%, depending on the fish species and its source. Thus, there is no combination of Great Lakes fish that would result in a nonhazardous diet to mink. The average allowable fish content in the mink diet for all of the Great Lakes fish was 7.5% (Giesy et al. 1994c).

The onset time and duration of exposure of mink to salmon is also problematic, such that mink could be eating large quantities of salmon for several months during sensitive reproductive periods. The late fall and early winter is a critical period for exposure to toxicants, when mink are mating and the females are pregnant. During this period, several species of anadromous salmonid fish migrate into Michigan rivers to spawn. The coho and chinook salmon die soon after spawning and are deposited on the shores of the river. With the onset of cold weather, the carcasses of the salmon can persist along the shore for a prolonged period of time. In this way, these fish could be a substantial source of contaminants to mink. For instance, using average concentrations of TEQs in fish from the Great Lakes, HQ values were all greater than 1.0, indicating some degree of risk to mink (Table 3-4). Most of the TEQs were contributed by PCBs.

From a time perspective, consuming chinook salmon for as little as 2 weeks could deliver the annual dose to mink that would be expected to affect reproduction (Giesy et al., 1994c).

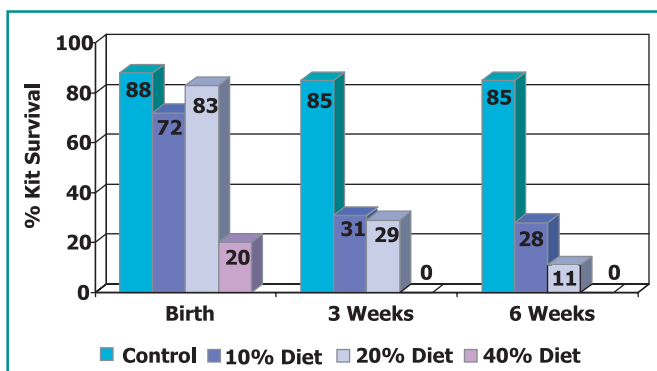


Figure 3-20. Survival of mink kits fed various proportions of Great Lakes fish in the diet (Tillitt et al. 1996; Heaton et al., 1995a).

Table 3-4. Hazard quotients for consumption of TEQ-containing Great Lakes fishes by mink

Common carp	47
Chinook salmon	11
Alewife	5.8
Northern pike	30

Calculated from information contained in Heaton et al. (1995a,b) and Tillitt et al. (1996), and unpublished data on concentrations of residues in fish tissue, J.P. Giesy.

Conclusions

- * All of the organochlorine compounds listed in the Stockholm Convention on POPs have been identified in fish and wildlife of the North American Great Lakes, even though some of these compounds, such as toxaphene, were never used in significant quantities in the region. This finding indicates that the source of some POPs in the Great Lakes ecosystem is long-range atmospheric transport.
- * Concentrations of POPs in Great Lakes fish have decreased significantly, approximately 25-fold, since the use of these compounds ceased within the Great Lakes basin.
- * Concentrations of the key POPs, such as *p,p'*-DDE and PCBs, in Great Lakes wildlife are currently either not declining or declining only slowly. It has been suggested that current concentrations of POPs are at a steady state, such that the amount lost to the sediments and exported either to the atmosphere or outflowing to the St. Lawrence River is balanced by ongoing input from rivers and the atmosphere. Thus, further input reductions of POPs and the continued recovery of wildlife populations depends, in part, on controlling long-range atmospheric transport of POPs from other parts of the world (Simcik et al., 1999).
- * Historically, several POPs accumulated to sufficient concentrations to cause adverse effects on fish and wildlife in the Great Lakes.
- * The degradation product of the organochlorine insecticide DDT, *p,p'*-DDE, which causes egg-shell thinning in raptors such as peregrine falcons, ospreys, and bald eagles, resulted in decreases in populations of these species to the point where they were almost completely extirpated from the Great Lakes basin.
- * PCDDs, PCDFs, and certain structurally similar PCBs caused a syndrome referred to as GLEMEDS in colonial fish-eating water birds of the Great Lakes. This syndrome, which is similar to chick edema disease, results in embryo lethality and developmental deformities in birds. The degree of expression of GLEMEDS has decreased as concentrations of PCDDs, PCDFs, and PCBs have declined, but certain species in some locations are still affected.
- * Concentrations of OC residues, primarily PCDD, PCDF, and PCBs, in fish are still sufficient to cause mortality in adult mink and severely reduce reproduction at as little as 1% Great Lakes fish in the diet.
- * Historically, concentrations of dioxin-TEQs, primarily from PCBs, were sufficient to cause blue-sac syndrome in sensitive fish species such as lake trout, and probably contributed to their population declines and restricted recovery. Currently, concentrations have decreased to a point where the incidence of blue-sac syndrome is rare.
- * At present, bald eagles nesting along the shorelines of the Great Lakes and along anadromous, accessible rivers have diminished reproductive capacity compared with those living at inland sites.
- * Even though concentrations of many of the POPs have decreased in Great Lakes wildlife subsequent to restriction of their use in the Great Lakes basin, some species of wildlife in some locations continue to be affected.
- * Failure to control sources of POPs outside the Great Lakes basin will limit the ability of the Great Lakes wildlife to recover.
- * The Great Lakes experience demonstrates that it is possible to sufficiently contaminate environments with residues to cause adverse effects. The experience in the Great Lakes is also one of hope, because controls on the production and release of POPs can result in reduced environmental concentrations and wildlife recovery.

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